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Part IV

Environmental Protection Agency

Carbon Monoxide; Proposed Revisions to the National Ambient Air Quality Standards and Announcement of Public Meetings

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 50

[FRL 1448-6 Docket No. OAQPS 79-7]

Carbon Monoxide; Proposed Revisions to the National Ambient Air Quality Standards

AGENCY: Environmental Protection Agency.

ACTION: Proposed rule.

SUMMARY: In accordance with Sections 108 and 109 of the Clean Air Act as amended, EPA has reviewed and revised the criteria upon which the existing primary and secondary carbon monoxide standards are based. The revised criteria are being published simultaneously with this notice of proposed rulemaking. The existing primary and secondary standards for carbon monoxide are both currently set at 9 parts per million (ppm), averaged over a period of 8 hours, and 35 ppm averaged over 1-hour, not to be exceeded more than once per year. As a result of the review and revision of the health criteria, EPA proposes to retain the existing primary 8-hour standard at 9 ppm and to lower the primary 1-hour standard to 25 ppm. The change in the 1-hour standard is being proposed because of the more rapid accumulation of blood carboxyhemoglobin in moderately exercising sensitive persons compared to resting individuals. The impact of exercise, which is greater for short-duration exposures, was not considered in the original standard. A secondary or welfare effects standard is not deemed appropriate for this pollutant because environmental effects have only been observed at relatively high carbon monoxide concentrations. Because CO concentrations of such magnitude rarely if ever occur in the ambient air, this notice proposes to rescind the existing secondary standard. Also proposed is a change to a statistical standard rather than the current deterministic standard; i.e., allowable exceedances will be stated as an expected value, not an explicit value. Furthermore, EPA proposes to use a daily interpretation for exceedance of the CO standard, so that exceedances will be determined on the basis of the number of days on which 1- or 8-hour average concentrations are above the standard levels.

During the period between this proposal and final promulgation of the standard, EPA will continue its examination of health and welfare effects information contained in the criteria document and will continue to seek to involve the the public, particularly the most directly affected parties, in the process of reaching a final decision on the air quality standard. One or more public meetings on this proposal will be held between proposal and promulgation and will be announced in a subsequent **Federal Register** notice.

DATE: Comments must be received by November 10,1980.

ADDRESSES: Submit Comments (duplicate copies are preferred) to: Central Docket Section (A-130), Environmental Protection Agency, Attn: Docket No. OAQPS 79-7, 401 M Street, SW., Washington, D.C. 20460.

Docket No. OAQPS 79-7, containing material relevant to this propsoed rulemaking, is located in the Central Docket Section of the U.S. Environmental Protection Agency, West Tower Lobby Gallery I, 401 M St., SW., Washington, D.C. The docket may be inspected between 8 A.M. and 4 P.M. on weekdays, and reasonable fee may be charged for copying.

Availability of Related Information

Documents generated as a part of this standard review such as the Control Techniques Document, Regulatory Analyses, Exposure Analysis, Environmental Impact Statement and Community Impact Statement can be obtained from: U.S. Environmental Protection Agency Library (MD-35), Research Triangle Park, N.C. 27711, Telephone (919) 541-2777 (FTS 629-2777).

The criteria document is available from: Environmental Criteria and Assessment Office (MD-52), U.S. Environmental Protection Agency, Research Triangle Park, N.C. 27711 (Attn: Dr. David McKee).

The criteria document may be requested by calling (919) 541-3746 (FTS 629-3746).

FOR FURTHER INFORMATION CONTACT: Mr. Michael Jones, Strategies and Air Standards Division (MD-12), Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, N.C. 27711, Telephone (919) 541-5231 (FTS 629-5231).

SUPPLEMENTARY INFORMATION:

Background

On April 30, 1971, the Environmental Protection Agency promulgated in the **Federal Register** (36 FR 8186) National Ambient Air Quality Standards for carbon monoxide. The scientific, technical, and medical basis for these standards is contained in the air quality criteria document for carbon monoxide published by the U.S. Department of Health, Education, and Welfare in March 1970. Both the primary and secondary standards were set at levels of 9 ppm, 8-hour average, and 35 ppm, 1-hour average, neither to be exceeded more than once per year. The preamble to the regulation stated:

> The national primary standard for carbon monoxide, proposed on January 30, 1971, was based on evidence that low levels of carboxyhemoglobin in human blood may be associated with impairment of ability to discriminate time intervals. In the comments, serious questions were raised about the soundness of this evidence. Extensive consideration was given to this matter. The conclusions reached were that the evidence regarding impaired time-interval discrimination had not been refuted and that a less restrictive national standard for carbon monoxide would therefore not provide the margin of safety which may be needed to protect the health of persons especially sensitive to the effects of elevated carboxyhemoglobin levels above 2 percent. It is the Administrator's judgment that attainment of the national standards for carbon monoxide will provide an adequate safety margin for protection of public health and will protect against known and anticipated adverse effects on public welfare.

The evidence cited above concerning impairment of ability to discriminate time intervals is based on work by Beard and Wertheim. As a result of the failure of several researchers to replicate the Beard and Wertheim results, the implications of this evidence for standard setting have changed since the original standard was promulgated. EPA's interpretation of this evidence is discussed elsewhere in this proposed regulation.

Carbon monoxide (CO) is a colorless, odorless, tasteless gas that is generally unreactive with other pollutants or constitutents found in the urban atmosphere. The primary sources of CO emissions are mobile sources, such as automobile, truck, and bus exhausts. Other sources of CO include stationary sources such as primary metal, carbon black, and maleic anhydride industrial facilities.

The reductions in CO emissions required by EPA standards are achieved through Federal and State programs which have been formalized in regulations promulgated under the Clean Air Act. The federal programs provide for reductions in emissions nationwide through the Federal Motor Vehicle Control Program, which involves controls for automobile, truck, bus, motorcycle, and aircraft emissions, and the development of New Source Performance Standards. The state programs provide for additional control

measures through State Implementation Plans in those areas of the country where the federal programs alone do not assure the attainment of air quality standards.

Legislative Requirements Affecting This Proposal

Two sections of the Clean Air Act govern the development of a National Ambient Air Quality Standard. Section 108 (42 U.S.C. 7408) instructs EPA to document the scientific basis (criteria) for the standard, and Section 109 (42 U.S.C. 7409) provides instructions on reviewing criteria and establishing standards.

Air quality criteria are required by Section 108(a)(2) to reflect accurately, upon issuance, the latest scientific information useful in indicating the kind and extent of all identifiable effects on public health or welfare that may be expected from the presence of the pollutant in the ambient air. If in the Administrator's judgment, revised criteria make appropriate the proposal of new or revised standards, such proposal is to occur simultaneously with issuance of the final criteria document.

The primary standard is defined in Section 109(b)(1) as that ambient air quality standard the attainment and maintenance of which in the judgment of the Administrator, based on the published criteria and allowing an adequate margin of safety, is requisite to protect the public health. The secondary standard, as defined in Section 109(b)(2), must specify a level of air quality the attainment and maintenance of which in the judgment of the Administrator, based on the published criteria, is requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of the pollutant in the ambient air. These adverse welfare effects which are defined in Section 302(h) of the Act include effects on soils, water, crops, vegetation, man-made materials, animals, weather, visibility, hazards to transportation, economic values, personal comfort and well-being, and other factors.

Section 109(b) of the Act specifies that National Ambient Air Quality Standards are to be based on scientific criteria relating to the level that should be attained to adequately protect public health and welfare. Considerations of cost of achieving those standards or the existence of technology to bring about needed reductions of emissions are not germane to such a determination, as the words of the Act and its legislative history clearly indicate. Section 109(d) directs the Administrator to complete a review of all existing standards and criteria before the end of 1980 and at 5-year intervals thereafter and to revise them in whatever manner that review determines is necessary.

Assuring attainment and maintenance of ambient air quality standards is the responsibility of the states. Under Section 110 of the Act, the States are to submit to EPA for approval State Implementation Plans (SIP's) that provide for the necessary legal requirements for sources of the relevant pollutant so as to demonstrate attainment and maintenance of the standards by certain deadlines. In the case of carbon monoxide, numerous urban areas have failed to attain the present standards. In 1977, Congress amended the Clean Air Act by inserting several sections, mostly found in Part D of Title I of the Act as amended, to intensify the effort to attain the CO standard. New deadlines were established, and the states were required to submit revised State Implementation Plans by January 1, 1979, for all "non-attainment areas."

If promulgated, the proposed changes to the CO standard will not modify the attainment dates specified in Section 172 of the Clean Air Act. As explained elsewhere in this preamble, both the 1-hour and 8-hour standards will be restated so that compliance with them will be determined statistically on the basis of a daily interpretation for exceedances. In addition, the numerical value of the 1-hour standard will be decreased.

An analysis of data available from the States indicates that, with regard to State Implementation Plan development under Part 51, the proposed CO standards should have little or no impact on the present attainment status designation of any area. Only 6 designated nonattainment areas in the country now exceed the 1-hour (35 ppm) standard, and all of these are included in the 146 designated nonattainment areas that now exceed the 8-hour (9 ppm) standard. There are no sites that currently exceed the proposed 1-hour (25 ppm) standard that do not also exceed the 8-hour (9 ppm) standard. Therefore, although EPA proposes to decrease the numerical value of the 1-hour standard, this action should not cause the designation of additional nonattainment areas. Since the proposed 1-hour standard (25 ppm) is not effectively as stringent as the 8-hour standard, the 8-hour existing standard, which has been the controlling standard in the past, will remain so, and the decrease of the 1-hour standard will not cause the designation of additional nonattainment areas. If a state already has an adequate plan to meet the existing CO standards, such a plan should be sufficient to meet the proposed standards. The proposed standards will not substantially affect New Source Review Requirements. For these reasons, the states will not be required to file revisions to their implementation plans.

Development of Air Quality Criteria and Summary of General Findings From Review of Air Quality Criteria for Carbon Monoxide

On December 1, 1978 (43 FR 56250), EPA announced that it was in the process of reviewing and updating the 1970 criteria document for carbon monoxide in accordance with provisions of Section 109(d)(1) of the Clean Air Act as amended. In the process of developing the revised criteria document, EPA has provided a number of opportunities for review and comment by organizations and individuals outside the Agency. Two drafts of the criteria document have been made available for external review, and EPA has received a total of approximately 30 written comments on these first and second drafts. Each draft was also the subject of a meeting of the Clean Air Scientific Advisory Committee (CASAC) of EPA's Science Advisory Board and the CASAC CO Subcommittee. Each of these meetings was open to the public, and a number of organizations and individuals have presented both critical review and new information for EPA's consideration.

On the basis of a careful review of scientific information contained in the criteria document[2], EPA prepared a staff paper[3] in which several key areas were identified as major factors to be considered in the possible revision of the CO standards; the Agency presented this paper at a public meeting of the CASAC. These findings, favorably reviewed by the CASAC, are summarized in the following paragraphs.

1. Carbon monoxide is a colorless, odorless gas that is toxic to mammals because of its strong tendency to combine with hemoglobin to form carboxyhemoglobin (COHb). Thus, the oxygen-carrying capacity of the blood is reduced since hemoglobin that has combined with CO in this manner is not available to transport oxygen; furthermore, the presence of COHb inhibits the release of oxygen from the remaining hemoglobin. The resultant hypoxemia (deficient oxygenation of the blood) can have detrimental affects on the cardiovascular, central nervous, pulmonary, and other body systems. The relatively small reductions in the ability of the blood to deliver oxygen to these systems at 5 to 10 percent COHb may be

quite critical for persons suffering from cardiovascular diseases or chronic obstructive lung disease.

2. The COHb in an individual's bloostream reflects input from two sources: Endogenous (produced by the metabolic breakdown of hemoglobin and other heme-containing materials) and exogenous (derived from the external environment). The physiologic norm for endogenous COHb levels has been estimated to be rougly in the range of 0.3 to 0.7 percent,[4] but endogenous production may be significantly increased for persons with hemolytic anemias, in women during pregnancy and during menstrual cycle, and in persons taking certain types of drugs. With these exceptions, however, increments significantly above the physiologic norm (i.e., above 1 percent) are assumed to have resulted from exogenous sources such as cigarette smoking (which can produce COHb levels ranging from 2 or 3 percent up to as high as 15 to 17 percent) or community air pollution.

3. Low-level CO exposures (producing group mean COHb levels in the range of 2.7 to 2.9 percent) have been shown to cause aggravation of cardiovascular disease. This aggravation consists of decreased time to onset of pain, both in persons with angina, and in persons with intermittent claudication (lameness) as a result of peripheral vascular disease,[5-7] and is considered to be an adverse effect. The criteria document states that the limited studies on patients having cardiovascular disease suggests that the critical level of COHb is 2.5 to 3.0 percent and the CASAC CO Subcommittee cited 2.7 to 3.0 percent COHb as the critical level at which adverse health effects do occur.[8] Both the critical range suggested by CASAC and that stated in the criteria document are in close agreement with the group mean values reported in the human studies (2.7 and 2.9) that show effects at the lowest COHb concentrations.[5-7]

4. Conflicting results have been obtained in community studies examining the relationship between CO exposure and mortality from myocardial infarction (heart attack) and sudden death due to arteriosclerotic heart disease. Consequently, the possibility of such an association remains in question, and more research is needed to clarify this issue.

5. Relatively low-level CO exposures have been reported to produce effects on the central nervous system such as decrements in vigilance (ability to detect small changes in one's environment that occur at unpredictable times). Effects of CO on vigilance are in considerable dispute, but some evidence suggests that, if relevant variables are controlled, CO exposures at a threshold level of about 4 to 6 percent COHb may produce decrements in vigilance. Decrements in visual function and sensitivity have been reported at COHb levels as low as 4 to 5 percent.

6. Another central nervous system effect, impairment in discrimination of time intervals, has been reported by Beard and Wertheim [1] to result from low-level CO exposures (estimated COHb levels of 2 to 3 percent). Their study provided the basis for the 1971 air quality standard for CO; however, the revised criteria documents states that considerable questions have been raised as to the validity of these observations, with several attempts to replicate these findings having been unsuccessful.

7. CO exposures above 5 percent COHb have been reported to produce an approximately linear decline in the maximal oxygen uptake of healthy individuals undergoing maximal exercise protocols of several minutes' duration. Persons with chronic obstructive pulmonary disease have been reported to exhibit decreased exercise performance following CO exposures producing a group mean COHb level of 4.1 percent.

8. In long-term exposures of pregnant animals to CO, fetal COHb levels have been shown to exceed maternal COHb levels significantly after an uptake lag of a few hours, and fetal elimination of CO upon cessation of exposure was slower than maternal elimination. In several experimental animal studies, pregnant females were exposed to CO; deleterious effects were in general reported in the offspring (e.g., reduced birthweight, increased newborn mortality, and lower behavioral activity levels) even when the mothers were not affected. In human studies, similar effects have been reported in children of mothers who smoked cigarettes during pregnancy. This evidence is discussed in greater detail in a subsequent section of this notice.

9. The only significant effect on vegetation reported for CO levels that approach those that might occur in the ambient air is an inhibition of nitrogen fixation by micro-organisms in the root nodules of legumes. Since the threshold reported for this effect (100 ppm for 3 to 34 days), is well above those CO levels occurring in the ambient environment, it is very unlikely that any damage will occur to vegetation as a result of CO air pollution. Furthermore, compliance with any primary standard likely to result from this rulemaking would insure protection against such damage.

Reported Human Health Effects Levels

Mechanism of CO Toxicity

EPA interpets the existing health effects evidence presented in the criteria document to indicate that the principal mechanism of CO toxicity is through hypoxemia (deficient oxygenation of the blood). This mechanism suggests that adverse effects on the body result from the strong affinity of blood hemoglobin for CO (over 200 times greater than for oxygen), which results in the formation of carboxyhemoglobin (COHb). Thus, the oxygen-carrying capacity of the blood is reduced since hemoglobin that has combined with CO in this manner is not available to transport oxygen; furthermore, the presence of COHb inhibits the release of oxygen from the remaining hemoglobin. Effects on the cardiovascular, central nervous, and pulmonary, and other systems are directly related to this reduction in the ability of the blood to deliver oxygen to these systems.

Some researchers have suggested an alternative mechanism for carbon monoxide toxicity that results from a blocking of the energy flow at the cellular level through the cytochrome system.[2] In discussing some of these studies, members of the CO Subcommittee of the Clean Air Scientific Advisory Committee (CASAC) [9] raised the possibility that the results found by these researchers could be more readily explained on the basis of the test protocols than as an alternative mechanism of toxicity. In several of these studies, animals were exposed to very high CO concentrations (greater than 100,000 ppm) for short periods of time, with the result that the animals died at a total-body COHb level lower than that which would be required if the CO dose had been administered as a lower concentration given over a longer period of time. The CASAC indicated that the hypoxemia mechanism probably provides an adequate explanation of this phenomenon as a manifestation of the "bolus effect", wherein a high concentration inhaled over a short period of time results in a portion of the blood supply that is essentially devoid of oxygen reaching the heart, with life-threatening consequences. Since the CASAC questioned the sufficiency of the information base on which the cytochrome system toxicity mechansim was proposed, EPA has decided to focus this NAAQS review on the hypoxemia mechanism and related COHb levels associated with observed adverse health effects.

Description of Adverse Effects

Cardiovascular System Effects. Angina pectoris is a cardiovascular disease in which mild exercise or excitement produces symptoms of pressure and pain in the chest because of insufficient oxygen supply to the heart muscle. Angina patients who have been exposed to low levels of CO while resting have subsequently exhibited, during exercise, reduced time to onset of angina. Increased duration of the angina attacks has also been reported.

EPA considers such aggravation of angina to be an adverse health effect for several reasons. First, it may result in cardiovascular damage, which is unquantifiable using present technology. Second, aggravation of angina may be the first in a series of progressively more serious symptoms that accompany cardiovascular disease. At low levels of oxygen deprivation, angina patients experience symptoms of chest pain described above. Coronary insufficiency is a more serious symptom that occurs at greater levels of oxygen deprivation. This symptom is sometimes accompanied by changes in enzyme levels and electrocardiographic irregularities. The most serious symptom in this continuum of effects is myocardial infarction. In addition to longer and more intense pain, myocardial infarction is accompanied by irreversible heart damage (death of myocardial cells) as indicated by enzyme level changes and electrocardiographic alterations. Finally, because aggravation of angina may be the first in a series of symptoms that may lead to permanent heart damage, EPA considers aggravation of angina an adverse effect and an indicator that more serious effects may occur in some individuals at the same COHb levels.

Aggravation of angina has been convincingly demonstrated by Anderson et al.[5] and Aronow and Isbell [6] at group mean COHb levels of 2.7 to 2.9 percent, which are lower than those associated with any other measurable adverse effect of CO exposure. In a 1978 study,[10] Aronow reported angina aggravation at COHb levels in the range of about 1.8 to 2.3 percent, but these COHb levels were obtained through a passive smoking exposure regime, with possible confounding factors. The appropriate utilization of this study in the standard-setting process will be discussed in a subsequent section of this notice.

Patients having peripheral vascular disease may also have this condition aggravated by CO exposure. In the one clinical study [7] in which such an effect was examined, 10 persons with intermittent claudication (lameness) due to occlusive arterial disease were exposed to 50 ppm of CO for 2 hours, with subsequent exercise until leg pain occurred. The COHb levels produced by this exposure (group mean of 2.8 percent) significantly decreased the time to onset of pain and cessation of activity.

Another cardiovascular system effect of concern is the possible detrimental effect of increased blood flow that occurs as a compensatory response to CO exposures. This response could result in coronary damage or other vascular effects due to the cardiovascular system being pushed beyond its capabilities. Cardiovascular damage and electrocardiogram abnormalities have been reported in persons who have experienced acute non-fatal CO poisoning (20 percent COHb) or who have been chronically exposed to CO in the workplace. Chronic exposure to average CO levels of 70 ppm, with peak levels of about 300 ppm (no COHb levels reported), has been associated with increased mortality from heart failure in a Japanese population.[11] Some epidemiological studies in Los Angeles [12–14] have suggested that increased mortality from myocardial infarction (death of heart tissue) may be associated with high ambient air concentrations of CO (estimated COHb levels in the range of 8 to 17 percent), but in a similar study Kuller et al.[15] failed to find such a correlation with considerably lower ambient CO levels. Radford [16] evaluated patients admitted to the myocardial infarction research unit at the Johns Hopkins University. While the investigator's diagnoses were consistent with both acute and chronic effects on the myocardium of long-term exposure to CO, the effects observed could not be clearly related to that factor. Therefore, the possibility of an association between CO levels in the ambient air and incidence of myocardial infarction or sudden deaths due to arteriosclerotic heart disease remains in question; more research is needed to clarify this issue.

In a recent epidemiology study, Kurt et al.[17] reported an increase in the number of cardio-respiratory complaints by patients at the emergency room of a Denver hospital on days when the 24-hour mean and maximum 1-hour mean ambient CO concentrations at a nearby monitoring site equalled or exceeded 5 and 11 ppm respectively, as compared to days when the corresponding concentrations were below those levels. In commenting on an early report of this study, however, the CASAC CO Subcommittee [8, 9] expressed the need for caution in interpreting this study because (1) the cardio-respiratory complaints evaluated in the study are inadequate indicators of cardiovascular disease aggravation, (2) the authors did not report any COHb levels for the patients evaluated and (3) the reported single monitoring site near the hospital is inadequate to determine the exposures sustained by the patients.

Central Nervous System Effects. Some studies [18–21] have reported that CO exposures resulting in COHb levels of 1.8–7.6 percent have produced decrements in vigilance (the ability to detect small changes in one's environment that take place at unpredictable times). The effects of CO on vigilance are in considerable dispute, however, since similar studies [22–27] have failed to observe similar effects. EPA's judgment of the evidence is that, if relevant variables are controlled, CO exposures at a threshold level of about 4–6 percent COHb may produce decrements in vigilance. Decrements in visual function and sensitivity have been reported at COHb levels as low as 4 to 5 percent.[28, 29]

EPA considers vigilance and visual function effects to be important since these functions are components of more complex tasks, such as driving, and reduced altertness or visual sensitivity could lead to increased accidents. Several studies discussed in the criteria document [2] suggested that elevated COHb levels adversely affect the performance of complex tasks, and suggestive (but not conclusive) evidence has been reported [30] indicating that a greater proportion of drivers in fatal accidents have COHb levels above 5 percent. Accordingly, the possibility of an interactive effect between alcohol and CO that has been suggested by some experimentation is of particular concern.

Pulmonary Function and Exercise Effects. In its discussion of several studies using sub-maximal exercise for short periods (5 to 60 minutes), the criteria document [2] states that oxygen uptake during work does not appear to be affected even when COHb levels are as high as 15 to 20 percent, although several of these studies have shown that these COHb levels produce increased heart rates. In maximal exercise protocols of several minutes' duration, COHb levels in the range of 5–33 percent have been demonstrated to produce a linear decline in maximal oxygen uptake (and hence work capacity).[2] MacMillan[31] reported that competitive swimmers have impaired performance after travelling to meets along a prescribed 2-hour route in

congested traffic, with average CO levels in expired air reaching 30 ppm (estimated COHb of about 6 percent). These results may, however, reflect interaction with other pollutants since ambient air exposures were examined in this study rather than systematic, controlled exposures to CO.

Although only a few studies have been conducted to examine the effects of CO exposure on persons with chronic obstructive pulmonary disease (e.g., asthma, emphysema, and chronic bronchitis), this group is presumably at high risk to CO exposures. A reduction in oxygen supply due to increased COHb levels could exacerbate existing effects of low oxygen levels caused by impaired respiratory system functioning. Aronow et al.[32] exposed 10 persons with chronic obstructive pulmonary disease (emphysema) to 100 ppm CO for 1 hour, producing a group mean of 4.1 percent COHb. Time to onset of marked dyspnea (difficulty in breathing) was reduced 33 percent during expercise as compared to COHb levels of 1.5 percent. The investigators concluded that the limited exercise performance after CO exposure was probably a cardiovascular limitation rather than a respiratory one.

Fetal Development. In several experimental animal studies that are discussed in the criteria document,[2] pregnant females were exposed to CO, and in general the offspring showed deleterious effects even when the mothers were not affected. For example, Astrup et al.[33] exposed pregnant rabbits to 90 ppm CO continuously for 30 days, with resultant maternal COHb levels of 9 to 10 percent. Birth weights decreased 11 percent and the newborn mortality rate increased to 10 percent from a control value of 4.5 percent. In another study,[34] the offspring of rats exposed to 150 ppm CO throughout gestation (maternal COHb levels of 15 percent) weighed slightly (3 percent) less at birth and failed to gain weight as rapidly after birth. Reduced brain protein levels at birth and lower behavioral activity levels through the preweaning period were observed. In many cases, the deleterious effects have been shown to disappear by adulthood, but the inference that such effects may occur in humans during maturation is of concern with respect to possible impacts on learning and social behavior development.

In the criteria document,[2] there is discussion of some experimental animal studies in which short-term maternal CO exposure resulted in lower COHb levels in the fetus than in the mother; however, in these studies, the fetus was still adversely affected. Longo and Hill[35] examined long-term maternal CO exposures and found that fetal COHb levels exceeded maternal values after an uptake lag of a few hours following initiation of exposure. At equilibrium, fetal COHb levels significantly exceeded maternal values, and fetal elimination of CO after cessation of exposure was slower than maternal elimination. The COHb concentration in human fetal blood has been reported[36] to vary from 0.7 to 2.5 percent (for non-smoking mothers), with the ratio of fetal to maternal COHb levels varying from 0.6 to 1.6.

The biologic effects of CO exposure on fetal tissues during intrauterine development require clarification. The ability of CO to decreased the oxygen transport capacity of maternal and fetal hemoglobin may result in interference in fetal tissue oxygenation during important developmental stages. Whereas a normal adult has reserve capacity and compensatory responses that enable him to handle moderately high COHb levels without irreversible consequences, the fetus may under normal conditions be operating close to critical levels in terms of tissue oxygen supply. Thus, even moderate CO exposures may have a deleterious effect on fetal development.

Some verification of this hypothesis has been suggested in studies examining the impact of maternal smoking on the unborn child, as well as by studies of the impact of altitude on the unborn child. Several studies of have demonstrated that babies born to smoking mothers have reduced birth weight, as do children born at higher altitudes. Some question remains as to the relationship of fetal deaths to maternal smoking, but, in several studies in which data from large population samples were used, investigators concluded that perinatal deaths of infants of mothers who smoke do increase when these data are corrected for other factors affecting perinatal death rates (e.g., maternal age, the number of children previously borne, race, and social status). The causes of this increased perinatal mortality have not been adequately identified.

The effect on surviving children of CO derived from maternal smoking is not well understood. Fedrick et al.[37] reported almost a twofold increase in the incidence of congenital heart disease in the infants of mothers who smoke. British studies[38,39] of large population groups have found highly significant differences in the attainment of reading skills at 7 years of age between the children of mothers who smoked and those who did not. In a follow-up study[40] of these children at 11 years of age, investigators found several months' retardation in general ability, reading, and mathematics in a comparison between the children of mothers who smoked (0.5 pack of cigarettes or more per day; average COHb levels, though not reported, may have been 3 to 4 percent or more) and children of those who did not. While the fact that cigarette smoke contains substances other than CO prevents a direct application of the results of these studies in setting the CO standard, the studies do suggest the need for caution in protecting unborn children from such potentially deleterious effects of CO exposures.

Population Groups Sensitive to Low Levels of CO

On the basis of the criteria document discussion of the principal adverse effects associated with low levels of CO exposure, EPA has concluded that the following groups may be particulary sensitive to exposures of CO: Angina patients, individuals with other types of cardiovascular disease, persons with chronic obstructive pulmonary disease, anemic individuals, fetuses, and pregnant women. While no evidence is presented in the criteria document that healthy children are particularly sensitive to CO, concern exists that they also may be at increased risk to CO exposure because of the increased oxygen requirements that result from their higher metabolism rates.

In EPA's judgment, the available health effects data identify persons with angina and those with other types of cardiovascular disease as the groups at greatest risk from low-level, ambient exposures to CO. The low threshold for persons with angina results from the fact that the angina condition is due to an insufficient oxygen supply to cardiac tissue, so that persons with this condition have an inadequate reserve capacity and an impaired ability to compensate for the effects of CO.

The second group of prime concern consists of individuals suffering from other cardiovascular diseases, such as peripheral vascular disease. While much less information is available concerning the effects of CO on this group, one clinical study by Aronow [7] showed significantly decreased exercise time to onset of leg pain at group mean COHb levels of 2.8 percent for persons with peripheral vascular disease. The two major types of peripheral vascular disease are arteriosclerosis and atherosclerosis. A 1972 National Health Interview Survey [41] estimates that 700,000 persons, or 0.34 percent of the total U.S. population, have

arteriosclerosis. Estimates for atherosclerosis and other types of peripheral vascular disease are not available.

The number of persons who had angina and other types of cardiovascular diseases was estimated by the U.S. National Health Examination Survey (NHES),[42] which during the period 1960 through 1962 examined a sample of the civilian non-institutionalized population of age 18 to 79 years; the purpose of the survey was to obtain information on the prevalence of cardiovascular and other diseases. This survey estimated that 4.5 million persons (4.0 percent of the adult population) had definite or suspected angina pectoris, either with or without myocardial infarction. Thus, angina patients represented a large fraction of the 5.5 million persons (5.0 percent of the adult population) estimated to have definite or suspected coronary heart disease; this category consisted almost entirely of angina and myocardial infarction. The survey also estimated the prevalence rates of definite or suspected coronary heart disease as a function of age; among persons over 55 years old, prevalence rates ranged between 10 and 17 percent for various 5-year age groupings.

In addition, a group of persons probably not identified in this survey would also seem to have cardiovascular problems that place them at risk to CO exposure. Approximately 400,000 persons die each year from sudden cardiac death; of these, nearly 25 percent have had no prior recognized symptoms of heart disease.[43] The results of one survey[44] indicated that nearly 40 percent of the victims of sudden cardiac death had consulted a physician during the month prior to death but had reported symptoms "so vague and inexplicit that coronary heart disease would have been difficult to infer." Therefore, the above NHES estimates may be somewhat low, since most of these persons without any evident symptoms of heart disease would probably not have been detected by the survey examinations.

On the basis of the available effects data, EPA is focusing on angina patients and those with other types of cardiovascular disease as the most sensitive groups. Other groups such as fetuses, anemics, and persons with chronic obstructive pulmonary disease can reasonably be projected to be affected at CO levels possible in ambient air exposures. Individuals with dietary or pathological anemia are probably at greater risk due to reduced oxygen-carrying capacity of their blood. A 1973 National Health Interview Survey[45] estimates that nearly 3.0 million persons (1.4 percent of the U.S. population) suffer from anemia. Persons with hemolytic anemia (who have increased hemoglobin destruction rates) would be particularly at risk because of their increased endogenous production of COHb. The Survey estimates of specific anemias having a hemolytic component range from 90,000 to 230,000 persons (0.04 to 0.11 percent of the U.S. population). Similarly, there are approximately 3.1 million live births per year in the United States or 1.5 percent of the population.[46] About 1 million pregnancies per year occur among women living in the central cities of metropolitan counties, where CO ambient levels tend to be greatest.[47] This latter group, therefore, would be at increased risk to CO exposure. Because of the lack of human data for non-cardiovascular disease groups, however, the potential effects on such persons will be considered in determining the margin of safety for the CO standard.

CO Exposure and Resulting COHb Levels

In developing the proposed standards, it is necessary to make judgments concerning the relationship between ambient carbon monoxide exposure and resulting COHb blood level. The primary factors determining the final level of COHb in individuals are the following: concentration of CO inhaled and duration of exposure, alveolar ventilation rates (which depend on the level of activity), initial COHb level, endogenous CO production, blood volume, hemoglobin concentration, relative affinity constant of hemoglobin for CO and oxygen (O_2), barometric pressure, and relative diffusive capability of the lungs.[48] EPA has chosen to exclude consideration of smokers from the following discussion since the primary source of CO and resulting COHb levels for these individuals is from the intake of tobacco smoke. The contribution of ambient CO exposures to the final COHb level of smokers is relatively insignificant.

When individuals inhale air containing a constant concentration of CO, several hours are required for them to reach a state of equilibrium, at which point the COHb level in the body no longer increases. For a given CO level, the time required to reach equilibrium is influenced by a number of factors, the most important for normal individuals being the level of activity as measured by the alveolar ventilation rate (V_A). At low levels of activity (rest/intermittent light activity, V_A=5–10 L/min), approximately 8–12 hours are needed for equilibrium to be achieved. For moderate walking (3 miles per hour, V_A=20 L/min), the equilibrium level of COHb may be reached in half that time, or around 4–6 hours.

Individuals are generally not, however, exposed to constant concentrations for long periods of time. The non-equilibrium levels of COHb must be determined in order to evaluate the COHb levels associated with alternative 1- and 8-hour average standard levels for CO. Thus, EPA must rely on a model or equation to obtain information on COHb levels resulting from short-term CO exposures.

A model known as the Coburn equation[48] has been developed which permits the calculation of COHb concentration as a function of time, with the physiological factors previously mentioned considered. Peterson and Stewart[49,50] have reported excellent correlation between COHb values measured in both male and female subjects and those predicted by the Coburn equation. In addition, at levels of activity sufficient to increase alveolar ventilation up to 2.5 times above resting levels, they measured COHb levels that were consistent with the theoretical values obtained by use of the Coburn equation. While further experimental verification is needed to demonstrate that the Coburn equation accurately predicts uptake and excretion of CO under a variety of conditions, EPA believes it is the best tool available for estimating the COHb levels that will result from short-term (1- to 8-hour) exposures to ambient CO concentrations. Furthermore, the CASAC agreed that for a given set of physiological factors, the Codburn model predicts with reasonable accuracy the typical COHb levels resulting from specified CO exposures for non-equilibrium situations.[51]

Table 1 presents baseline or nominal estimates of the Coburn model as to the COHb levels expected to be achieved by non-smokers exposed to various constant concentrations of CO for either 1 or 8 hours. This table does not take into account the impact of fluctuating CO concentrations on CO uptake; this impact will be discussed in a subsequent section. A moderate lung ventilation rate, equivalent to a 3 mile-per-hour walk, has been selected as a reasonable estimate of the mean ventilation achieved by individuals with angina or coronary heart disease while exercising. The impact of activity level on rates of COHb accumulation is most apparent for shorter duration exposures. As illustrated in the table, very little difference exists in the COHb levels for

resting and moderately exercising persons for 8-hour exposures to CO concentrations near the current standard level. Consequently, assumptions regarding exercise levels at the 8-hour averaging time are not as critical in estimating COHb levels as for the 1-hour averaging time.

Table 1.—*Predicted COHb Response to Exposure to Constant CO Concentrations*

[Percent COHb based on Coburn Equyation [1]]

CO (ppm)	Exposure time (hours)			
	1		8	
	Intermittent rest/light activity	Moderate exercise	Intermittent rest/light activity	Moderate exercise
7.0	0.7	0.7	1.2	1.2
9.0	.7	.8	1.4	1.5
12.0	8	1.0	1.8	1.9
15.0	1.0	1.1	2.2	2.4
20.0	1.1	1.4	2.9	3.1
25.0	1.3	1.6	3.6	3.8
35.0	1.6	2.1	4.9	5.3
50.0	2.2	2.9	7.0	7.6

[1] Assumed parameters: V_A (Alveolar ventilation rates): intermittent rest/light activity=10/min, moderate activity=20 L/min (equivalent to 3 mph walk on level ground or light industry or housework); Hb (hemoglobin)=15 g/dL (normal male); altitude=sea level; initial COHb level=0.5 percent (normal level due to endogenous CO production); V_{CO} (endogenous CO production rate)=0.007 mL/min (normal); V_b (blood volume)=5500 mL (normal male); M (relative affinity constant of hemoglobin for CO and O_2)=218; D_L (lung diffusivity for CO)=30 mL/(min-mm Hg) (nominal).

Increased exodgenous CO accumulation rates or endogenous CO production, or both, could result in COHb levels higher than intlicated in Table 1 for individuals with anemia, pregnant women, and individuals taking certain tyupes of medication or drugs. For example, because of their reduced hemoglobin (Hb) levels, anemic individuals approach equilibrium levels of COHb more rapidly than those with normal Hb levels, and consequently attain a higher COHb level for a given exposure to CO. Such considerations in selecting a standard with an adequate margin of safety will be discussed below.

Selecting the Level of the Primary Standard

Section 109(b)(1) of the Clean Air Act requires EPA to set primary standards, based on the air quality criteria and allowing an adequate margin of safety, which in the Administrator's judgment are requisite to protect the public health. The legislative history of the Act makes quite clear the Congressional intent to protect sensitive persons who in the normal course of daily activity are exposed to the ambient environment. Air quality standards are to be established with reference to protecting the health of a representative, statistically related, sample of persons comprising the sensitive group rather than a single person in such a group.

EPA's objective in setting the standard levels for the carbon monoxide primary standard is, therefore, to select levels that will reflect an accurate consideration of the existing medical evidence, an adequate assessment of the uncertainties in this evidence, and a reasonable provision for scientific and medical knowledge yet to be acquired, so as to protect sensitive population groups with an adequate margin of safety. The criteria document supports the conclusion that a clear threshold of adverse health effects cannot be identified with certainty for CO or even for COHb levels, which are more directly related to health effects than ambient concentations of CO. Focusing on COHb levels, there is rather a continuum consisting of COHb at which health effects are certain through levels at which scientists can generally agree that health effects have been convincingly shown, and down to levels at which the indications of health effects are less certain and harder to identify. (This does not necessarily mean that there is no threshold, other than zero, for CO; it simply means that no clear threshold can be identified with certainty based on existing medical evidence.) Thus, selecting a standard that takes into account the known continuum of effects is a judgment of prudent public health practice, and does not imply some discrete or exact margin of safety appended to a known threshold.

The uncertainties associated with such a judgment relate to several factors. First, human susceptibility to health effects varies considerably among individuals in the population and EPA cannot be certain that experimental evidence has accounted for the full range of susceptibility. Second, EPA cannot be certain that all effects occurring at low CO levels have been identified or demonstrated. Third, there is variability and uncertainty about the relationship between ambient CO concentrations and elevated COHb levels for different segments of the population and for varying exposure conditions. These and similar sources of uncertainty are discussed more fully below.

The Clean Air Act, as the Administrator interprets it, does not permit him to take factors such as cost of attainment or attainability into account in setting the level of the standard; the standard is to be one that will adequately protect public health. Actual implementation of the standards is reserved primarily to the States under Section 110 of the Act, and therefore a certain degree of flexibility is accorded individual States in determining how to attain the standards in the most economical and practical matter. The Administrator recognizes, however, that controlling CO to very low levels is a task that can have significant impact on economic and social activity.

This recognition causes the Administrator to reject as an option the setting of a zero-level standard as an expedient way of protecting public health without having to decide among uncertainties. Whether or not that option is available under the law, EPA believes that Congress did not intend the Administrator to "take the easy way out" simply because the health evidence supporting a standard above zero is beset with uncertainties. Rather, the uncertainties must be confronted and taken into account. Thus, the decision as to what standard protects public health with an adequate margin of safety must be based on a consideration of the uncertainty that any given level is low enough to prevent adverse health effects and on the relative acceptability of various degrees of uncertainty, given the seriousness of the effects.

In selecting the proper levels for the standard, EPA must make assessments and judgments in the following areas:

1. Identification of the lowest COHb level that medical research to date has convincingly linked to adverse health effects in sensitive persons.
2. Description of the nature and severity of effects against which the standard is to protect.
3. Characterization of the uncertainties in the medical evidence concerning the lowest COHb levels at which sensitive individuals would be affected.
4. Characterization of the sensitive population.
5. Selection of a CO standard level that provides an adequate margin of safety, based on COHb values that could be achieved by sensitive population groups and taking into account the various uncertainties involved.

Reported Effect Levels and Severity of Effects

Table 2 provides a summary of key health effects studies reporting effects due to elevated blood COHb levels. The table is included only as an aid for the following discussion and should be used only in conjunction with qualifying statements made in the criteria document regarding the technical merits

of each study. While the table includes studies reporting effects in two of the basic categories described earlier, the selection of a standard will be based primarily on the cardiovascular effect category, as discussed previously.

Table 2.—*Key Studies (Compilation of Effects Reported in Selected Human Studies Examining Carbon Monoxide Exposures)*

CO concentration, ppm	Exposure duration, hours	Percent carboxyhemoglobin, group mean (and range)	Reported effect(s) [1]	Reference(s)
Not reported (passive smoking).	2	1.8 and 2.3	Duration of exercise until onset of angina decreased 22 and 38 percent.	9. Aronow, 1978.
50	2	2.7 (2.5 to 3.0)	Duration of exercise until onset of angina decreased 16 percent.	Aronow and Isbell, 1973.
50	2	2.8 (2.4 to 3.1)	Duration of exercise until onset of leg pain (intermittent claudication) in persons with peripheral vascular disease decreased 17 percent..	7. Aronow et al., 1974.
50	[2]4	2.9 (1.3 to 3.8)	Duration of exercise until onset of angina decreased 15 percent.	5. Anderson et al., 1973.
150	[3]	3.9 (3.4 to 4.4)	Impairment of ventricular (heart) functioning in anginal patients with coronary heart disease.	51. Aronow et al., 1974.
100	1	4.0 (3.0 to 4.9)	Duration of maximal exercise in 10 healthy nonsmokers decreased 5 percent, with 1 subject exhibiting an electrocardiographic abnormality following CO and exercise.	52. Aronow and Cassidy, 1975.
100	1	4.1	Duration of exercise until marked difficulty in breathing (dyspnea) in persons with emphysema decreased 33 percent.	31. Aronow et al., 1977.
100	[2]4	4.5 (2.8 to 5.4)	Duration of exercise until onset of angina decreased 15 percent and duration of pain increased 31 percent.	5. Anderson et al., 1973.
42 to 63 (mean 53)	[4]1.5	5.1 (3.8 to 8.0)	Duration of exercise until onset of angina decreased 30 percent after freeway exposure and was still decreased 15 percent 2 hours later.	53. Aronow et al., 1972.
2	[5]2	2.9 (0.6 to 3.9)		

[1] Percentages represent the group mean response.
[2] Intermittent.
[3] Not reported.
[4] Freeway.
[5] After return from freewawy.

The present NAAQS for CO is not based on the cardiovascular effect category but, rather, on central nervous system effects. The **Federal Register** notice (36 FR 8186) promulgating the existing standard identified 2 percent as the critical COHb level on the basis of a study by Beard and Wertheim [1] which reported an impairment in discrimination of time intervals in subjects having estimated COHb levels of 2 to 3 percent. The revised criteria document states that considerable questions have been raised as to the validity of these observations and points out that attempts to replicate these findings have been less than satisfactory. As stated previously, EPA has concluded that other central nervous system effects, such as vigilance and visual function decrements, may occur in the range of 4 to 6 percent COHb.

EPA proposes to base its selection of the standard on the evidence regarding the aggravation of angina and other cardiovasular diseases by CO exposures. As discussed previously, EPA considers such aggravation to be an adverse health effect. This effect appears to be more serious than the time discrimination effect on which the present standard is based. Such cardiovascular effects, as exhibited by reduced exercise time to onset of chest pain (angina) or leg pain (peripheral vascular disease), have been reported in three studies at 2- to 4-hour CO exposures that result in group mean COHb levels in the range of 2.7 to 2.9 percent.[5-7] The CASAC CO Subcommittee [8] also suggested that the medical evidence to date best supports the 2.7 to 3.0 percent COHb range as the lowest group mean level convincingly linked to adverse health effects in sensitive persons.

In all three studies, individuals were affected at COHb levels below 2.7 percent, as low as 2.4 percent in one study.[7] In the study by Anderson et al.[5], one individual showed a slight (3 percent) reduction in time to onset of angina at a final COHb level of 1.3 percent. This individual's reported initial COHb value of 0.0 percent, which seems unrealistically low and probably reflects the uncertainty involved in measuring very low COHb levels, raises some question about the validity of this data point.

In 1978, Aronow [10] reported that a lower range (1.8 to 2.3 percent) of group mean COHb resulted in angina aggravation, but these COHb levels were obtained through a passive smoking exposure regime, with possible confounding factors. The criteria document states that "it is possible that in addition to carbon monoxide and nicotine, other components of tobacco smoke, including oxides of nitrogen and hydrogen cyanide, and possibly psychological factors, may have contributed to the decrease in exercise performance." The CO Subcommittee of the CASAC [8] agreed and cautioned EPA not to use this study as the basis for identifying the lowest COHb level that medical research to date has convincingly linked to adverse health effects in sensitive persons. On the basis of these comments, EPA is considering the 1978 Aronow study only as a factor in selecting an adequate margin of safety. In a subsequent section of this preamble, a new human CO exposure study conducted by Dr. Aronow, not available at the time of criteria document publication, is discussed.

Uncertainty Concerning Lowest Effect Levels

While the lowest group mean COHb level convincingly linked to adverse health effects is in the range of 2.7 to 2.9 percent, a clear threshold for adverse health effects has not been established. Several factors make it difficult, if not impossible, to identify the minimum level of COHb associated with adverse health effects. As indicated above, human susceptibility to health effects varies considerably among individuals, and EPA cannot be certain that experimental evidence has accounted for the full range of susceptibility. Indeed, for ethical reasons, clinical investigators have generally excluded from study individuals who may be very sensitive to CO exposures, such as those with myocardial infarction or multiple disease states (e.g., angina and hemolytic anemia). Another factor is that there is an absence of negative clinical CO studies at lower COHb levels; in fact, no clinical studies have been reported for COHb levels lower than those reported in Table 2. In this regard, EPA has funded additional research that will examine aggravation of angina at lower COHb levels than previously tested. Finally, EPA cannot be certain that it has identified all adverse health effects that are related to lower level CO exposures.

Potentially Sensitive Populations

Convincing experimental evidence indicates that persons with angina and other cardiovascular diseases are sensitive to low-level CO exposures. In addition, theoretical considerations, animal studies, and evidence from studies of cigarette smokers provide reason to believe that other groups in the population (e.g., fetuses, anemics and visitors to high altitudes) may be sensitive to CO exposure. However, since no human experimental evidence exists that identifies CO effect levels for these other groups, EPA is considering the possible effects of CO on these groups only in its determination of an adequate margin of safety.

1. *Fetuses.* One important area in which animal studies provide information and in which no human data exist is in the effects of CO on the fetus. Studies have shown that, for long-term CO exposures, the animal fetus develops higher COHb concentrations than their pregnant mothers. Detrimental effects on fetal development (reduced birth weight, increased newborn mortality, and behavioral effects) have been observed in experimental animal CO exposure studies and in studies of the effects of maternal smoking on human fetuses. These findings denote a need for caution in assessing possible human fetal effects from CO exposures and in evaluating the margin of safety provided by the CO standard.

2. *Anemic Individuals.* Because of their reduced hemoglobin levels, persons with anemia are expected to attain higher COHb levels than a normal person for a given short-term CO exposure, or attain equilibrium COHb levels more quickly. In addition, certain types of anemia (hemolytic anemia) result in an increased endogenous production of CO, so that increased COHb levels would be expected for a given exogenous CO exposure. Consequently, anemic individuals who also suffer from angina or who are pregnant would seem to represent particularly high-risk categories. Since, however, very little data are available regarding the effects of CO exposure on anemic individuals, EPA is considering such persons only in its determination of an adequate margin of safety.

3. *Visitors to High Altitude Locations.* Hypoxemia can occur at higher altitudes as a result of reduced oxygen pressure in the atmosphere; in addition, an increase in altitude can increase the rate of COHb accumulation in the blood. Normal residents of high-altitude locations appear to have adjusted to the elevation and do not seem to exhibit an interaction between the effects of altitude and CO exposures [55] although the possibility of adverse effects on residents is a factor in setting a standard with an adequate margin of safety. Of greater concern to EPA are those persons with impaired cardiovascular systems who visit high altitude locations and may be adversely affected by the combination of altitude and CO hypoxemia.

CO Uptake Uncertainty

1. *Individual Variation and Dynamic Considerations.* Table 1 presents baseline estimates of the Coburn model as to the COHb levels expected to be achieved by non-smokers exposed to constant CO concentrations for 1 or 8 hours. Uncertainties exist, however, in using these estimates to decide which standard provides an adequate margin of safety. First, even among normal persons variations exist in the physiological parameters used in the Coburn model. These variations are sufficient to produce noticeable deviations from the COHb levels in Table 1 that were predicted using baseline or nominal parameters. Even more substantial variations in these parameters are observed in population groups (such as anemics) that may, for this reason, be more sensitive to CO exposure. Second, predictions based on exposure to constant CO concentrations inadequately represent the response of individuals exposed to widely fluctuating concentrations of CO that typically occur in ambient exposure situations. Ott and Mage [56] investigated this point using an empirically derived CO uptake model to estimate COHb responses as a function of time for actual 1-hour average CO air quality data measured over a 12-month period in San Jose, California. Their calculations indicated that the assumption of a constant CO concentration for an 8-hour averaging time tends to underestimate the COHb response to CO concentrations that are actually fluctuating on a much shorter time scale.

EPA has attempted to represent these uncertainties by preparing a sensitivity analysis using the Coburn model to predict the COHb responses to specific patterns of fluctuating CO concentrations for individuals with physiological parameters different from the baseline values. As explained in a staff analysis paper that has been placed in the docket (OAQPS 79–7, II–A–1), twenty different sets of actual 1-hour average CO air quality data were adjusted to simulate attainment of alternative 8-hour standards. For each of these air quality patterns, the COHb response was evaluated on an hour-by-hour basis by using the Coburn model prediction of the COHb resulting from one hour's exposure as the initial COHb level for the next hour.

Table 3 presents the results of this dynamic simulation of COHb responses to a range of realistic patterns of fluctuating CO concentrations. The baseline case is included for comparison with the estimates given in Table 1 for exposure to constant CO concentrations. In Case 2, the physiological parameters of the individuals all differ from baseline values in the direction that increases the COHb response to CO exposures. The parameters used for this case do not seem unreasonable, since the value of each parameter lies within the range reported in the literature as normal. The choice of these parameters is explained in the staff paper mentioned above (OAQPS 79–7, II–A–1). Case 3 is an individual with hemolytic anemia; the physiological parameters chosen for this case are average values reported for 12 hemolytic anemia patients evaluated by Coburn et al. [48]

Table 3.—*Coburn Model Estimates of COHb Levels Associated With Attainment of Alternative 8-Hour CO Standard Levels*[1,2]

Standard level, ppm	Maximum COHb levels (percent) predicted on day when annual maximum 8-hour CO concentration occurs, for a range of actual air quality patterns adjusted to simulate attainment of the specified standard		
	Case 1[3]	Case 2[4]	Case 3[5]
7	1.2–1.5	1.6–2.0	1.8–2.2
9	1.4–1.9	2.0–2.6	2.2–2.8
12	1.8–2.4	2.5–3.3	2.8–3.6
15	2.3–3.0	3.0–4.1	3.3–4.4

[1] COHb responses to fluctuating CO concentrations were dynamically evaluated using the Coburn model prediction of the COHb resulting from one hour's exposure as the initial COHb level for the next hour.

[2] Coburn model parameters:
Case 1: As given in table 1, except only $V_A = 10$ L/min.
Case 2: $V_A = 10$ L/min; Hb = 13 g/dL; altitude = sea level; initial COHb = 0.7 percent; $V_{co} = 0.014$ mL/min; $V_b = 3500$ mL; $D_{Lco} = 40$ mL/(min-mm Hg); M = 246.
Case 3: $V_A = 10$ L/min; Hb = 9 g/dL; altitude = sea level; initial COHb level = 1.9 percent; 0.030 mL/min; $V_b = 4600$ ml; $D_{Lco} = 25$ mL/(min-mm Hg); M = 246.

[3] Baseline or nominal physiological parameters.

[4] High rate of physiological parameters for normal persons (sea level).

[5] Typical hemolytic anemic individual.

It would be desirable to conduct a sensitivity analysis similar to that reported in Table 3 for alternative 1-hour average standards. Since, however, EPA does not have CO air quality data for periods of less than 1-hour, it is not possible to perform a dynamic simulation of COHb responses to CO concentrations that are fluctuating on a time scale less than 1 hour. Consequently, EPA has conducted a sensitivity analysis that simply depicts the variation in estimated COHb responses to the maximum 1-hour CO

concentrations permitted by alternative standards for individuals with physiological parameters different from the baseline values. Table 4 presents the results of this analysis for each of the three cases illustrated in Table 3 and the case where an individual has an initial elevated COHb level due to exogenous sources (e.g., passive smoking).

While the upper end of the range of COHb estimates in Tables 3 and 4 indicates the potential that some individuals might achieve COHb levels in the vicinity of the reported effects levels, these tables do not provide information on the possible number of individuals at various COHb levels due to variations in physiological parameters. EPA undertook an analysis to obtain such information, and the results are described in the above mentioned analysis paper (OAQPS 79–7, II–A–1). The analysis assumes that all of the physiological parameters used in the Coburn equation are normally distributed in the sensitive population, with the exception of the alveolar ventilation rate (exercise level). The analysis of alternative 8-hour standards is based on an alveolar ventilation rate of 10 liters per minute (rest/intermittent light activity). For the analysis of alternative 1-hour standards, the alveolar ventilation rate was set at 20 liters per minute (moderate exercise).

With respect to Case 3, EPA estimates the number of cardiovascular and peripheral vascular disease patients who also have hemolytic anemia probably is less than 1,000, compared with an estimated 7,700,000 cardiovascular and peripheral vascular disease patients (based on 1979 population data). This estimate is based on the fact that most cardiovascular patients are over age 40 and on the fact that the vast majority of hemolytic anemic individuals are under 40. Because the group of individuals with cardiovascular or peripheral vascular disease who also have hemolytic anemia is so small, EPA is focusing its decision on protecting the 7,700,000 sensitive individuals with cardiovascular heart or peripheral vascular disease. This reflects EPA's response to the legislative history which calls for the Administrator to use his judgment in identifying and protecting the most sensitive group of citizens but not necessarily the most sensitive individuals in that group.

Table 4.—*Coburn Model Estimates of COHb Levels Associated With Alternative 1-Hour CO Standard Levels*

Standard level	COHb levels (percent) predicted for a range of ventilation rates for exposure to the maximum 1-hour average CO concentration permitted by the specified standard [1]			
	Case 1 [2]	Case 2 [3]	Case 3 [4]	Case 4 [5]
20	1.1–1.4	1.8–2.2	2.7–2.9	2.3–2.6
25	1.3–1.6	2.1–2.6	3.0–3.3	2.6–3.0
35	1.6–2.1	2.7–3.5	3.6–4.1	3.2–3.9

[1] Coburn model parameters are as presented for Table 3, except a range of alveolar ventilation rates is presented. The lower value represents rest/intermittent light activity (V_A=10 L/min) and the higher value represents moderate exercise (V_A=20 L/min).
[2] Baseline or nominal physiological parameters.
[3] High range of physiological parameters for normal persons (sea level).
[4] Typical hemolytic anemic.
[5] Same as case 2, except assumes initial elevated COHb (1.5%) from external sources.

Case 2 is based on individuals with physiological parameters whose values are in the high range for normal persons, but does not provide information on the number of individuals in the sensitive population that would attain those levels. Table 5 shows the estimated percentage of the sensitive population with COHb greater than, or equal to, the specified final COHb value. It was assumed that the sensitive population was exposed to air quality levels associated with alternative standards shown in the table. In the analysis, twenty different sets of actual 1-hour average CO air quality data were adjusted to simulate attainment of alternative 8-hour standards. The estimates in Table 5 are based on a representative pattern from the group of twenty air quality data sets. The sensitive population considered is that segment of the population having cardiovascular heart disease (CHD) or peripheral vascular disease. The total estimated population in this sensitive group in 1979 was 7,700,000 people. One point to keep in mind is that the estimates shown in Table 5 are based on the assumptions that the entire sensitive population is exposed to air quality levels just meeting the standard and to the representative pattern of 1-hour average CO concentrations. In actuality, only a fraction of the population would be exposed under these conditions and, thus, the number of individuals at high COHb levels would probably be smaller than the estimates given in Table 5.

Table 5.—*Cumulative Percent of Sensitive Persons With COHb > Specified Final COHb Value, When Exposed to Air Quality Associated With Alternative Standards*

[Cardiovascular heart and peripheral vascular disease] [1]

	Dynamic calculation [3]			Static calculation [4]		
	7 ppm, 8 hr	9 ppm, 8 hr	12 ppm, 8 hr	20 ppm, 1 hr	25 ppm, 1 hr	30 ppm, 1 hr
Final COHb (percent): [2]						
2.9			0.01			<0.01
2.7			.2			.01
2.5			6		<0.01	.2
2.3		<0.01	43		.01	4
2.1		[illegible]	[illegible]	<0.01	.6	29
1.9	<0.01	13	[illegible]	.1	13	73
1.7	.2	73	100	5	61	[illegible]
1.5	20	[illegible]	100	[illegible]	[illegible]	100
1.3	[illegible]	100	100	[illegible]	100	100

[1] Estimated total cardiovascular heart disease and peripheral vascular disease population is 7,700,000.
[2] Assumes all physiological inputs to Coburn equation are normally distributed in the sensitive population.
[3] COHb responses to fluctuating CO concentrations were dynamically evaluated using the Coburn model prediction of the COHb resulting from 1 hour's exposure as the initial COHb level for the next hour. The series of 1-hour CO concentrations used was a representative pattern selected from 20 sets of actual air quality data that just reached the level of the specified 8-hour standard. These results are based on alveolar ventilation rate of 10 L/min (intermittent rest/light activity).
[4] COHb levels calculated on the basis of constant level exposure and an alveolar ventilation rate of 20 L/min (moderate activity).

The results of the analysis indicate that more than 99 percent of the sensitive group (i.e., those individuals with cardiovascular heart or peripheral vascular disease) would not be expected to reach, or exceed, 2.1 percent COHb upon exposure to air quality associated with the proposed standard of 9 ppm, averaged over 8 hours. Approximately 13 percent of the sensitive group could be expected to reach or exceed 1.9 percent COHb if they were exposed to CO levels associated with the proposed 9 ppm, 8-hour standard. The proposed standard of 25 ppm, averaged over 1 hour, provides an approximately equivalent degree of protection.

Case 4 in Table 4 shows that quite high COHb levels are theoretically possible, given a high initial COHb from external sources. As in the above analysis, EPA has estimated how many people are expected to reach such final

COHb levels. The estimates are presented in Table 5. In the analysis, it was assumed that the entire population was given the previous exposure and that the entire population was exposed to alternative 1-hour air quality standard levels. While it is theoretically possible to reach a final COHb of 3.0 percent (see Table 4) for a 25 ppm standard, we have estimated (Docket Reference OAQPS 79–7, II–A–1) that only a very small fraction of the sensitive group (less than .001 percent) would ever experience such an outcome even if these worst-case assumptions are used.

2. *Bolus Effect.* Another factor to be considered in selcting a standard with an adequate margin of safety is the uncertainty relating to adverse health impacts from very short-duration (5 to 10 minutes or less) high-level CO exposures. While the data base is incomplete regarding the mechanism or effect of the peak short-term exposures, the criteria document and the CASAC CO Subcommittee have emphasized the potential seriousness of CO delivered to the body in this manner. The consequence may well be adverse effects separate and, perhaps, more intense than those associated with elevated COHb concentrations, a phenomenon referred to as the bolus effect. While this factor should be considered in establishing an adequate margin of safety for the standard, existing air quality data do indicate that attainment of a longer averaging time standard should limit the magnitude of short-term peak concentrations, as discussed in a subsequent section of this notice. Consequently, the bolus effect, while of concern in selecting a margin of safety, does not appear to be an overriding consideration in this standard selection process.

Pollutant Interactions and Smokers

Few studies have examined the interactions of carbon monoxide with other pollutants. One investigation [57] examining simultaneous exposures of normal and reactive subjects to 0.25 ppm ozone, 0.3 ppm nitrogen dioxide, and 30 ppm CO for 2 hours on 2 successive days detected no consistent lung function changes attributable to exposure. The angina aggravation observed in Aronow's passive smoking study [10] may have been due to interactions between carbon monoxide and other pollutants (e.g., nicotine, hydrogen cyanide, particulates, and nitrogen oxides) contained in the tobacco smoke. The possible interactions between CO and smoke-related pollutants should be considered in determining a margin of safety for the CO standard since persons may, in addition to ambient CO exposures, be exposed in passive smoking situations.

On the other hand, EPA does not propose to consider active smokers in determining the need for a margin of safety for the CO ambient air standard. Little or no evidence exists that would suggest the need for more restrictive national ambient air quality standards to protect smokers from a possible incremental COHb burden from the air. In fact, even in a pristine environment, smokers will have COHb levels ranging from 2 to 17 percent. Furthermore, existing evidence suggests that cigarette smokers, exposed to an environment where CO is at the ambient standard level, are generally exhaling more CO into the air than they are inhaling from that environment. While these individuals are at a definite risk from accelerated incidence of heart and related disease, these risks are principally associated with cigarette smoking, which is generally voluntary, and not with incremental CO levels at or near the standard level.

Choice of the Standard Levels

As discussed above, in EPA's determination of an air quality standard with an adequate margin of safety, the Agency must consider uncertainties regarding the lowest effect levels for aggravation of angina and other cardiovascular diseases and peripheral vascular disease, as well as uncertainties in the uptake of CO by sensitive population groups in its determination of an air quality standard with an adequate margin of safety. Several studies [4–6] have reported aggravation of angina and intermittent claudication by CO exposures producing group mean COHb levels in the range of 2.7 to 2.9 percent, with some individuals exhibiting aggravation as low as 2.4 to 2.5 percent. The sensitivity analysis conducted by EPA indicates that more than 99 percent of the sensitive population (those who suffer from cardiovascular heart or peripheral vascular disease) would be kept below 2.1 percent COHb upon exposure to air quality associated with the standard of 9 ppm, averaged over 8 hours. On the basis of these considerations, EPA proposes to set the 8-hour average National Ambient Air Quality Standard for carbon monoxide at 9 ppm. This standard will be attained when the expected number of days per calendar year with maximum 8-hour average concentrations above 9 ppm is equal to or less than 1, as explained in a subsequent section of this notice.

With regard to the 1-hour average CO standard, EPA has previously recognized that, in setting the existing 35 ppm standard, the Agency failed to consider the influence of light exercise on the aggravation of angina patients' condition when exposed to CO.[58] Tables 1 and 4 illustrate the impact of moderate exercise (walking 3 miles per hour) on uptake of various constant CO concentrations. The CASAC CO Subcommittee indicated that this exercise level represented a reasonable estimate of the mean ventilation rate achieved by individuals with angina and other cardiovascular diseases while exercising.[51]

Comparison of the COHb values in the moderate exercise column of Table 1 for 1-hour CO exposures to the values given for exposure to 9 ppm for 8 hours indicates that a 25 ppm 1-hour average standard would be comparable to the proposed 8-hour standard in terms of COHb protection. The results of the sensitivity analysis shown in Table 5 also indicate that a 25 ppm 1-hour standard would prevent more than 99 percent of the sensitive population from attaining 2.1 percent COHb upon exposure to 25 ppm, averaged over 1 hour. This protection is approximately the same as that provided by the proposed standard of 9 ppm, averaged over 8 hours. The National Academy of Sciences has recommended a 1-hour CO standard approximately 30 percent more stringent than the present (35 ppm) standard.[59] On the basis of the preceding analyses and considerations, EPA proposes to revise the 1-hour standard from 35 to 25 ppm.

Given the various uncertainties referred to above, the COHb levels associated with the 9 ppm, 8-hour and 25 ppm, 1-hour standards do not afford a large degree of protection for sensitive persons exposed at these ambient concentrations. EPA is therefore soliciting comments from the public as to whether the 1-hour and 8-hour standards provide an adequate margin of safety.

In determining the appropriate margin of safety, the Agency has considered the following factors, all of which have been discussed previously and are briefly restated below in no particular order of significance:

(1) The Aronow passsive smoking study,[12] which may indicate a lower threshold of adverse health effects from COHb in spite of the possibly confounding presence of other components of tobacco smoke.

(2) Various uncertainties regarding the lowest adverse health effect levels from CO, including those related to (a) variations in individual susceptibilities; (b) the lack of clinical studies, because of ethical considerations, on very sensitive groups such as those who have

suffered myocardial infarctions, and multiple diseased patients such as those with both angina and hemolytic anemia; (c) the absence of negative clinical studies in the range of lower CO levels; and (d) whether EPA has in fact identified all adverse health effects that are related to low-level CO exposures.

(3) Uncertainties regarding additional potentially sensitive groups, who may have elevated COHb levels because of increased exogenous accumulation rates or endogenous production. These potentially more sensitive groups include (a) fetuses, (b) pregnant women (particularly in the central cities), (c) anemics, (d) visitors to high altitude locations, and (e) persons on certain medications.

(4) Uncertainties regarding the uptake of CO, including those related to (a) the accuracy of the Coburn equation in assessing variations in the population, (b) whether predictions based on exposures to constant levels of CO adequately reflect the widely fluctuating concentrations that occur in ambient exposure situations, and (c) the expected number of sensitive individuals at various COHb levels after exposure to air quality levels associated with alternative standards.

(5) Uncertainty regarding adverse health effects that may result from very short duration high-level CO exposures (the bolus effect).

(6) Possible interactions between CO and other pollutants, especially those encountered in passive smoking situations.

Other Aspects of the Primary Standard

Averaging Time

Selection of the averaging time, form of the standard, and allowed number of exceedances are all key elements in the process of reaching a decision on the CO standard and have a direct effect on the required level of control. The NAAQS's for CO are presently 9 ppm and 35 ppm for 8-hour and 1-hour averaging times, respectively, not to be exceeded more than once per year.

The original 8-hour averaging time was selected primarily because most individuals achieve equilibrium or near-equilibrium levels of COHb after 8 hours of exposure. As mentioned previously, approximately 4–12 hours are required to achieve an equilibrium level of COHb upon continuous exposure to CO. The time to reach equilibrium is influenced primarily by the exercise level of the individual, with shorter times required for greater exercise. Another basis for the 8-hour averaging time is that most people are exposed in approximately 8-hour blocks of time (e.g., work, sleep). With respect to the 1-hour averaging time, the health effects rationale is now stronger than in 1971. Aronow and his coworkers have conducted several studies in which health effects have been reported for persons with cardiovascular disease or chronic obstructive pulmonary disease after 1- to 2-hour exposures. In light of the above considerations, EPA does not propose to change the current 1-hour and 8-hour averaging times for the CO NAAQS. EPA is, however, soliciting public comment as to the necessity of maintaining two separate averaging times for the CO standard.

While the majority of health effects evidence is linked to 1 hour or longer exposures, there is also concern for shorter CO exposure durations. Exposure to much higher CO concentrations (200–2000 ppm) for very short periods of time (a few minutes) may result in significant localized COHb blood levels (bolus effect). Analyses of existing air quality data suggest, however, that attainment of a longer averaging-time standard will limit the magnitude of short-term peak concentrations in the ambient air. For example, air quality data obtained in 1974–1976 at a site in Los Angeles that is 6 meters (m) high and 3 m from the curb of a highway bearing an average traffic load of 25,000 vehicles per day were analyzed to determine, for various averaging times, the peak concentrations that would be expected if the present NAAQS's were just attained. The maximum values actually observed were 46 and 30 ppm for 1- and 8-hour averaging times. Using a two-parameter averaging time model,[60] EPA calculated that attainment of the 8-hour standard would result in peak 5- and 10-minute concentrations of 36 and 29 ppm, respectively. Although this site might not represent the worst possible situation, the analysis does seem to indicate that attainment of longer averaging time standards would tend to limit short-term peaks to levels that would not present a concern with regard to a bolus effect.

Form of the Standard

The current CO standards specify that the hourly average CO concentration must not exceed 40 mg/m^3 (approximately 35 ppm) more than once per year and that the 8-hour average CO concentration must not exceed 10 mg/m^3 (approximately 9 ppm) more than once per year. The limitations of this deterministic (once-per-year) approach were discussed in connection with the proposal of a revised ozone standard (43 FR 26967). A main concern with the present "once-per-year" form of the CO standards is that to maintain such a standard year after year necessitates a zero probability that the second high value will ever exceed the level of the standard. On a practical basis, however, permitting a single exceedance in a year means that there is some possibility of occasionally having two or more exceedances in a particular year. To resolve this logical conflict and to adjust for the effect of missing data, the ozone standard was proposed and promulgated in terms of the expected number of exceedances per calendar year (43 FR 26970, 44 FR 8220). EPA proposes to use this expected exceedances format for the CO standards.

EPA also proposes to use a daily interpretation for exceedances of the CO standard. This approach is consistent with the interpretation of exceedances for the ozone standard (44 FR 8220) and means that exceedances would be counted on the basis of the number of days on which the standard level was exceeded. As in the case of the ozone standards, the use of a daily interpretation is not predicated on the interpretation of the health data. At the same time, the health data do not argue against use of a daily interpretation. Use of such an interpretation, however, constitutes a data reduction step that simplifies daily reporting and reduces the possible impact of a single unusual meteorological event. Thus, exceedances of the 1-hour CO standard will be based upon the number of days with maximum hourly CO averages above the level of the 1-hour standard. The 8-hour standard will also be interpreted in terms of the number of days with 8-hour averages above the level of the 8-hour standard. This topic is treated in more detail in the following discussion of running averages.

Running Averages

The 8-hour CO standard warrants a more detailed discussion because of the complications associated with running averages. Because individual hourly values are available, it is possible to compute an 8-hour average beginning (or ending) with each hour of the year. This fact means that successive 8-hour averages will have 7 hourly values in common. The current CO standard was not explicit on how exceedances of the 8-hour standard level should be counted (36 FR 8187), although the subject of interpreting air quality data was subsequently discussed by EPA.[61]

To avoid any amgibuity in counting exceedances for the 8-hour CO standard and to clarify the computational procedures, EPA proposes that the CO standard include a specific method for

use with the 8-hour standard. For each hour of the day, there is an 8-hour average that ends at that hour (e.g., for 4:00 p.m. there is an 8-hour average covering the time period 8:01 a.m.–4:00 p.m.). For each day, the highest of these twenty-four 8-hour averages would be considered the daily maximum 8-hour average. If this value exceeds the level of the standard, then an exceedance has occurred on that day. It should be noted that with this method, there is the possibility that two daily exceedances could have common hourly values. For example, the 8-hour average covering 4:01 p.m.–midnight would be assigned to 1 day while the next 8-hour average, from 5:01 p.m.–1:00 a.m., would be assigned to the following day. A third daily exceedance, however, would always be on a third day and, thus, could not overlap either of the other 8-hour averages. In view of the expected exceedances format, this method appears to be a simple and adequate one for handling running averages.

Other alternatives were considered and should be briefly mentioned. One approach is to use all possible 8-hour averages for the year and an hourly interpretation so that more than one exceedance could be counted per day. This approach may be viewed as more consistent with the dosage received by a receptor, but is more stringent than the current form in that exceedances would not have to be non-overlapping. At the extreme, this interpretation could result in eight consecutive exceedances being counted from 8-hour averages that all overlap; therefore, this option was not chosen.

EPA also considered alternatives that would eliminate the possibility of overlapping exceedances by restricting the number of 8-hour averages to only three non-overlapping time periods in a day (e.g., 0:01 a.m.–8:00 a.m., 8:01 a.m.–4:00 p.m., and 4:01 p.m.–midnight). There is no assurance, however, that the maximum 8-hour average will occur during these time periods rather than one of the other twenty-one 8-hour periods that are not used. Another possible option is to select the daily maximum 8-hour average only from those 8-hour periods that are entirely contained within that calendar day. There are seventeen such 8-hour time periods covering the successive intervals: 9:01 a.m.–8:00 a.m., 1:01 a.m.–9:00 a.m., * * *, 4:01 p.m.–midnight. This particular scheme can miss peak 8-hour averages that occur late in the evening (e.g., 5:01 p.m.–1:00 a.m.).

Because of meteorological factors, high 8-hour averages can occur in time periods extending from the evening rush hour through the early morning hours (e.g., 5:01 p.m.–1:00 a.m., 6:01 p.m.–2:00 a.m.). This problem can be alleviated by offsetting the time periods considered so that the daily maximum is selected from the following seventeen 8-hour periods: 3:01 a.m.–11:00 a.m., 4:01 a.m.–noon, * * *, 7:01 p.m.–3:00 a.m. In this approach, the majority of the hours in each 8-hour time period all come from the same day. With this offset, 8-hour time periods from the evening rush hour through the early morning hours are covered. It should be noted that in each of the alternative approaches that would result in non-overlapping exceedances, every hour of the day is included in at least one 8-hour average that is used but there are certain 8-hour periods that are not covered. Comments are invited on the form of the standard and the method for handling running 8-hour averages.

Definition of When the Standard is Attained

EPA proposes to add Appendix I to 40 CFR Part 50 to interpret the standard and to explain how to determine when the standard is or is not being attained. Although this appendix is conceptually identical to Appendix H of the ozone standard (44 FR 8220), there are certain computational differences. In particular, no allowance is made for seasonal monitoring for carbon monoxide and no exclusion of missing values is allowed on the basis of adjacent daily values. The basic adjustment for missing data for CO is to assume that the fraction of missing daily values that would have exceeded the standard level is the same as the fraction of measured values that exceeded the standard level. While ambient CO levels are generally seasonal, the nature of CO emissions makes it possible to exceed the standard level at any time of the year, and therefore EPA does not intend to allow seasonal exclusions for the CO attainment computations.

Significant Harm

Section 303 of the Clean Air Act authorizes the Administrator of EPA to identify, for ambient air populations, exposure levels that constitute "an imminent and substantial endangerment to the health of persons." This language provides the basis for the "significant harm level" referred to in the following discussion.

In commenting on the authority provided in Section 303, the Senate Committee on Public Works [62] stated that

> "emergency authority is necessary to provide for immediate, effective action whenever air pollution agents reach levels of concentration that are associated with (1) the production of significant health effects, (2) incapacitating body damage, or (3) irreversible body damage in any significant portion of the general population. The term 'significant portion' (was) not intended to exclude sensitive elements of society such as asthmatics, but only those groups of particularly susceptible persons for whom other precautionary measures should be taken. Secondly, the emergency situation exists whenever there is any perceptible increase in the mortality rate."

EPA's regulations (40 CFR 51.16) require the adoption by States of contingency plans to prevent ambient pollutant concentrations from reaching specified significant harm levels. The existing significant harm levels for CO were established in 1971 (36 FR 24002) at the following levels and averaging times:

50 ppm—8-hour average
75 ppm—4-hour average
125 ppm—1-hour average

These designations were associated with a 5 to 10 percent blood COHb concentration as the critical range to be avoided. On the basis of EPA's reassessment of the earlier data and assessment of more recent medical evidence, no modifications are being proposed to the existing significant harm designations. EPA has assessed the medical evidence on exposure to higher CO concentrations that could lead to significant harm and has placed this assessment in the docket (OAQPS 79–7) for this proposed rulemaking.

Welfare Effects and the Secondary Standard

Carbon monoxide is a normal constituent of the plant environment; plants can both metabolize and produce CO. This fact may explain the relatively high levels of CO necessary to produce detrimental effects to vegetation. The lowest threshold for significant effects in vegetation has been reported at 100 ppm for 3 to 34 days for an inhibition of nitrogen fixation in legumes. Since CO concentrations of this magnitude are rarely if ever observed in the ambient air, it is very unlikely that any damage to the environment will occur from CO air pollution. Because no standard appears to be requisite to protect the public welfare from any known or anticipated adverse effects from ambient CO exposures, EPA is proposing to rescind the existing secondary standard.

Regulatory, Environmental, and Exposure Impacts

As has been noted, the Clean Air Act specifically requires that National Ambient Air Quality Standards be based on scientific criteria relating to the level that should be attained to protect public health and welfare

adequately. EPA interprets the act as excluding any consideration of the cost or feasibility of achieving such a standard in determining the level of the ambient standards. However, in compliance with the requirements of Executive Orders 11821 and 11949 and Office of Management and Budget Circular A–107 and with the provisions of the recently issued Executive Order 12044, EPA has analyzed economic impacts associated with efforts to attain these standards.

Carbon monoxide air pollution is a problem afflicting most urban areas. Mobile sources are the primary cause of the problem; motor vehicles account for nearly 75 percent of the nationwide CO emissions and most high CO monitor readings occur in areas with heavy traffic concentration. Thus, control strategies for attaining the CO ambient air quality standard will have to focus on reducing the emissions from motor vehicles.

Many areas will not be able to attain the CO standard by 1982 and will, therefore, have to institute control strategies for mobile sources. Foremost among these control strategies will be inspection/maintenance (I/M) programs, which will be required in areas that cannot demonstrate attainment by 1982. Transportation control measures (TCM's) which reduce vehicle miles travelled and improve traffic flow to alleviate "hot spot" problems, will also be required in several areas with the most severe problems.

The number of areas in which the proposed standards will not be attained by 1987 depends on a number of factors, including the effectiveness of emission control systems, the effectiveness of I/M programs, growth of vehicle miles travelled (VMT) in urban areas, traffic patterns, and actual air quality values once all monitors meet EPA's siting, instrumentation, and quality assurance criteria. Taking into account all these factors, the number of non-attainment areas in 1987 (assuming no change in the standards in the interim) could range from 2 areas with the most severe problems up to possibly 20 areas. The net cost of implementing I/M programs and TCM's for CO control in response to the proposed standards will amount to approximately $400 million per year, which is in addition to about $2.4 billion required for the CO portion of the Federal Motor Vehicle Control Program.

The CO control measures should have a favorable impact on energy consumption, the savings from which are reflected in the cost given above. I/M programs will possibly have an appreciable impact on fuel economy of repaired vehicles, while TCM's will reduce fuel consumption by reducing miles travelled and improving traffic flow.

Environmental impacts associated with control of CO emissions have been examined in a document available in docket number OAQPS 79–7. This environmental impact analysis indicates that control strategies required to attain the standard will have minimal adverse impacts on other environmental media.

Finally, EPA has prepared an exposure impact analysis document, which has been placed in docket number OAQPS 79–7. This document provides estimates of the number of person-hours of exposure to CO concentrations in excess of specified values for alternative levels of the standard; these estimates are furnished on a nationwide basis and for specific cities.

Copies of the documents presenting EPA's regulatory analyses in support of the CO standard, the environmental impact statement, and the exposure analysis are available from the U.S. EPA Library at the address given earlier in the Dates and Addresses section of this notice.

Revisions to Part 50 Regulations

In proposing to revise the CO standard, it is necessary for EPA to add Part 50 Appendix I, "Interpretation of the National Ambient Air Quality Standard for Carbon Monoxide"; the reason is that additional guidance is necessary to understand the statistical nature of the revised standard.

Part 51 Regulations and SIP Development

This proposed rule will not result in modification to the existing Part 51 regulations. With regard to SIP development under Part 51, the proposed CO standards should have little impact on the attainment status designation of most areas. However, where sufficient data is available to support a change in designation, either the State or EPA may initiate such a change under the terms of Section 107 of the Clean Air Act after the standard is promulgated. While unlikely, the proposed standard may impact the control strategies needed in some areas. These impacts will need to be analyzed on a case-by-case basis and EPA will provide guidance on this matter when the standards are promulgated. The standards will not substantially affect New Source Review Requirements.

Federal Reference Method

The measurement principle and calibration procedure applicable to reference methods for measuring ambient carbon monoxide concentrations to determine compliance with the standard are not affected by this rulemaking. The measurement principle and the current calibration procedure are set forth in Appendix C of 40 CFR Part 50. Reference methods—as well as equivalent methods—for monitoring carbon monoxide are designated in accordance with 40 CFR Part 53. A list of all methods designated by EPA as reference or equivalent methods for measuring carbon monoxide is available from any EPA Regional Office, or from EPA, Department E (MD–76), Research Triangle Park, N.C. 27711.

Relationship to Existing Monitoring Regulations

The specific requirements for carbon monoxide (CO) monitoring are established in 40 CFR Part 58. These regulations were promulgated on May 10, 1979 (44 FR 27558), and represent significant improvements over the November 1971 SIP monitoring regulations. In particular, the monitoring deficiencies that Part 58 is designed to correct are: untimely or incomplete reporting of monitoring data, collected data of unknown or questionable quality, lack of uniformity in station location, probe siting and related sampling and analysis methods, and the absence of quality assurance programs. The old regulations provided for fixed sites only, and the number of sites was based on population and degree of pollution. Under the new provisions, fixed and movable sites are allowed, but the number and location of stations are determined only after a detailed and comprehensive analysis of air monitoring objectives. Other specific changes made to improve the regulations require the adherence to specified network design and siting criteria and the development and implementation of a quality assurance program.

Under the regulations promulgated on May 10, 1979, each State is required to design a network to measure ambient concentrations of all pollutants for which standards have been established under 40 CFR Part 50. Within each State, the stations making up such networks are to be designated as the State and Local Air Monitoring Stations (SLAMS). The States are responsible for consulting with the Regional Administrator during the network design process to ensure that the design criteria for SLAMS contained in Appendix D to Part 58 are met. At the present time, drafts of these network descriptions are undergoing review by the Regional Offices. The final network design for each State will

be subject to the approval of the Regional Administrator.

A critical subset of the SLAMS network will be designated as National Air Monitoring Stations (NAMS). For CO, the stations designated as NAMS are needed to assess the overall air quality progress resulting from the emission controls required by the Federal Motor Vehicle Control Program (FMVCP). Two CO NAMS will be required in each urbanized area greater than 500,000 population, with one of these stations located in a peak concentration area such as near major traffic arteries or near heavily travelled streets in downtown areas (microscale). The second CO NAMS will be a neighborhood station and should be located in areas with a stable, high population density and having a high traffic density. Micro-scale stations will generally characterize the peak concentrations usually found near heavily travelled downtown streets (street canyons) and should be representative of several similar source configurations in the urban area. Neighborhood stations should represent the concentration levels which exist over a significant portion of the urban area.

As a part of its assessment of the impact of revising the 1-hour standard, EPA examined the ambient air quality monitoring, data reporting and surveillance provisions promulgated on May 10, 1979 (44 FR 27558) to determine if changes were needed. Based on data available from the States, no revisions to these regulations are warranted. If future ambient data, new sources, changes in emission patterns or other factors should indicate the need for more intensive monitoring in selected areas, the need for revision of the State networks can be identified during the annual review of the SLAMS network required by 40 CFR Part 58, § 58.20.

For additional details on network design and siting, see Appendices D and E of Part 58.

Public Participation

EPA has solicited public comment and critique on draft revisions to the criteria document as well as comments on a draft staff paper [3] that contained a preliminary assessment of the health effects evidence. Comments on the two draft revisions of the criteria document have been considered in the final document, published simultaneously with this proposal. An explanation of how EPA addressed these comments has been placed in docket number OAQPS 79–7.

On May 29, 1979, EPA announced in the **Federal Register** (44 FR 30760) a public meeting of the Science Advisory Board's Clean Air Scientific Advisory Committee (CASAC) to discuss the second draft of the revised criteria document and a draft staff paper [3] describing the major factos that EPA staff recommended be considered in the possible revision of the primary standards. In that notice, EPA also announced the availability of the CO staff paper. The staff paper: (1) Evaluated the critical health effects evidence; (2) described the most sensitive population groups; (3) described the relationship between CO exposure and resulting COHb levels; (4) recommended redefining the standard in a statistical, rather than deterministic, form; and (5) identified either 2.5–3.0 percent COHb or 1.8 percent COHb as the lowest convincingly demonstrated effects level, the lower level being dependent on use of the 1978 Aronow passive smoking study. The CASAC public meeting was held June 14–June 16, 1979; a transcript of the meeting is available in docket number OAQPS 79–7. During the meeting and afterwards, comments were received on the aforementioned issues as well as other topics related to the standard. These comments are summarized below and have been considered in the development of this proposed rulemaking. Because of possible confounding factors such as nicotine and particulates found in cigarette smoke, the CASAC [6] and some industrial groups have strongly advised EPA not use the 1978 Aronow study in selecting the lowest convincingly demonstrated effects level. The CO Subcommittee and CASAC as a whole, with one dissenting opinion, agreed that decreased time to onset of angina was an adverse health effect that had been shown to occur at group mean COHb levels in the range of 2.7 percent to 3.0 percent.[5]

The area of greatest controversy raised during the public review process concerns the significance of the effects reported at COHb levels below 3.0 percent to 4.0 percent. Several automobile manufacturers have maintained that decreased time to onset of pain for angina patients does not necessarily represent an adverse health effect. These groups argue that low-level CO studies have failed to show statistically significant changes in other clinical measures of cardiac function such as systolic time internals. The automotive groups have also stated that other factors such as cold weather, eating a heavy meal, or sitting next to a cigarette smoker may have a greater effect on an individual with angina than ambient CO exposures. As discussed elsewhere in this premable, EPA and the CASAC CO Subcommittee [6] do not agree with this judgment about the significance of the effects demonstrated for individuals with angina or other coronary artery or peripheral vascular disease conditions.

Several industrial groups have noted that effects reported in short-term, high-level exposures may not be the same as effects resulting from long-term, low-level exposures, even if the final COHb levels are identical. While EPA agrees that the manner in which dose is delivered may have an impact on the effects experienced, the Agency does believe that there is currently insufficient evidence to support an alternative mechanism of toxicity for standard-setting purposes.

The exclusion of smokers in selecting a standard with an adequate margin of safety was supprted by CASAC.[6] The CASAC also agreed that, on the basis of available health effects evidence, persons with angina, coronary artery, and peripheral vascular disease were the population groups at greatest risk to low-level ambient CO exposures.[6] Concern was expressed by CASAC that some sensitive population groups might be adversely affected as a result of the combined effects of hypoxemia due to high-altitude exposure and CO exposures. The CASAC stated that this factor should be considered in selecting a CO standard with an adequate margin of safety.[6]

The use of the Coburn model to determine COHb levels resulting from various CO exposures received little criticism during the public review process. CASAC indicated that the Coburn model was the best available tool for predicting COHb levels resulting from CO exposures.[6] The CASAC and other groups have generally supported redefining the standard in a statistical, rather than deterministic, form. The CASAC endorsed EPA's proposal to retain the 1-hour and 8-hour averaging times for the standard.[6] Some industrial groups have commented that the 1-hour standard could be dropped since the 8-hour standard was the controlling one in terms of stringency.

A recent study brought to EPA's attention (Case et al., 1979) [63] suggests that nitrogen oxides may impair oxygen transport through formation of nitrosyl hemoglobin (NOHb) and methemoglobin (MetHb), a form of hemoglobin that is incapable of combining with oxygen. The authors suggest that this mechanism operates in conjunction with both ambient and clinical exposures to CO and, thus, puts, into question the findings of studies described in the

criteria document. The Case study also questios the validity of COHb measurements made with instruments used in these studies.

On the basis of careful review of the Case article and consultation with the principal author, EPA does not concur with the written views of the author. There is no sound evidence available to document: (1) The extent of nitrosyl hemoglobin (NOHb) or methemoglobin (MetHb) formation either directly or indirectly from nitrogen oxides, (2) the relative accumulation of NOHb and MetHb from various media inputs, (3) the combined impact of COHb and MetHb or NOHb, and (4) the reversibility of MetHb formation relative to COHb. Furthermore, several prominent investigators in the field have expressed their view to EPA that the levels of MetHb and NOHb are negligible in persons exposed to ambient air and to atmospheres in clinical test chambers. EPA does, however, plan additional research in this area and encourages further comment on this topic during the development of this rule.

With regard to the validity of the COHb measurement technique, the statements made in the Case article were apparently based on analysis of an earlier spectrophotometric instrument. In the clinical studies cited in support of the proposed standard, however, the more recently developed CO-Oximeter instrument, which is reported to be accurate to within approximately ±0.1–0.2 percent, was used. Consequently, the above criticism appears to be inapplicable with regard to the accuracy of the reported COHb values.

EPA has recently received an unpublished draft copy of a new carbon monoxide human exposure study conducted by Dr. Wilbert Aronow. In this EPA sponsored study, Dr. Aronow reports a statistically significant decrease in the time to onset of angina for subjects exposed to 50 ppm CO for 1 hour compared to subjects exposed to compressed air. On a group mean basis the final COHb was 2.0 percent after exposure to 50 ppm CO for 1 hour compared with an initial mean COHb value of 1.1 percent prior to CO exposure.

The draft Aronow study has been submitted for publication in a peer-reviewed journal. EPA's current view is that the study should not be considered in this rulemaking, except perhaps as a factor in establishing a margin of safety, unless it has first been peer reviewed within the scientific community and also judged significant to warrant supplementation of the criteria document. EPA is therefore placing the study in the docket (OAQPS–79–7 II–A–2), submitting a copy to CASAC for its review, and is soliciting comments from the general public on the study and the role, if any, it should play in the final rulemaking.

Dated: July 31, 1980.

Douglas M. Costle,

Administrator.

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EPA proposes to amend Part 50 of Chapter I, Title 40, of the *Code of Federal Regulations* as follows:

§ 50.8 [Revised]

1. Section 50.8 is revised to read as follows:

The levels of the national primary ambient air quality standards for carbon monoxide, measured by a reference method based on Appendix C to this part and designated in accordance with Part 53 of this chapter, or by an equivalent method, are 25 parts per million (28.63 mg/m^3) for a 1-hour average concentration and 9 parts per million (10.31 mg/m^3) for an 8-hour average concentration. The standards are attained when the expected number of days per calendar year as determined by Appendix I to this part:

(a) With maximum 1-hour average concentrations above 25 parts per million (28.63 mg/m^3) is equal to or less than 1, and

(b) With maximum 8-hour average concentrations above 9 parts per million (10.31 mg/m^3) is equal to or less than 1.

Appendix I—Interpretation of the Nation Ambient Air Quality Standard for Carbon Monoxide

I. General

This appendix explains the necessary computations for analyzing carbon

monoxide (CO) data with respect to the 1-hour and 8-hour standards. The term "exceedance" is used throughout this appendix to describe a daily maximum value above the level of the standard. For the 1-hour standard, an exceedance refers to a daily maximum hourly value above the level of the 1-hour CO standard. For the 8-hour standard, an exceedance refers to a daily maximum 8-hour average above the level of the 8-hour CO standard. A CO value is said to be an exceedance if, after being rounded to the nearest ppm, it is greater than the level of the standard (fractional parts equal to 0.5 should be rounded up).

In general, the average number of exceedances per year must be less than or equal to 1 for each standard. In its simplest form, the number of exceedances at a monitoring site would be recorded for each calendar year and, then, average over the previous calendar years to determine if this average is less than or equal to 1. If data are missing, however, it is necessary first to compute the estimated number of exceedances for each calendar year on the basis of the observed number of exceedances plus an adjustment to account for missing values. The expected number of exceedances is then determined on the basis of the average of these annual estimates.

II. Data Handling Conventions

In order to compute the expected number of exceedances, it is necessary to define what is meant by a valid daily maximum value. It is assumed that hourly average carbon monoxide (CO) measurements are available on a clock-hour basis. The end point of the time period determines the day to which the value is assigned. Time periods ending at midnight are the last time periods of the day. For example, an 8-hour average from 4:01 p.m. to midnight is the last 8-hour average from 5:01 p.m. to 1:00 a.m. is the first 8-hour average of one day, while the 8-hour average of the next day. By this convention, the value is assigned to a day on the basis of when the receptor has accumulated the exposure.

A daily maximum hourly value is the highest of the 24 possible hourly averages for the day. This daily mixiumum hourly value is termed "valid" if at least 75 percent of the possible hourly averages were available or if at least one hourly average in the day exceeded the level of the 1-hour standard.

An 8-hour average is considered to be valid if at least 75 percent of the hourly averages for the 8-hour period were available. In the event that only six (or seven) hourly averages were available, the 8-hour average is computed on the basis of the hours available using six (or seven) as the divisor. Hourly values less than the minimum detectable limit should be replaced by one-half the minimum detectable limit for these computations. To ensure that potentially high 8-hour averages are not ignored, an 8-hour average with less than six hourly values will also be considered valid if substitution of one-half the minimum detectable limit for the missing values (and with 8 used as the divisor) results in an average above the level of the 8-hour standard. A daily maximum 8-hour average is the highest of the 24 possible 8-hour averages ending in that day. This daily maximum 8-hour average is considered to be valid if at least 75 percent of the 8-hour averages were valid or if there is at least one valid 8-hour average above the level of the 8-hour standard.

As a final convention, hourly values from the end of 1 year need not be carried over to compute 8-hour averages at the start of the next year. This convention should eliminate potential data processing problems with archived data.

III. Estimating the Number of Exceedances Expected in a Year

Because of practical considerations, a valid daily maximum hourly or 8-hour average may not exist for each day of the year. To account for the possible effect of missing data, an adjustment must be made to the number of exceedances in a calendar year. In this adjustment, the assumption is made that the fraction of mission values that would have exceeded the standard level is identical to the fraction of measured values above this level. This computation is to be made for all National Air Monitoring Stations (NAMS), State and Local Air Monitoring Stations (SLAMS), and all other sites that are scheduled to monitor for an entire year. Because of possible seasonal imbalance, this computation does not apply to Special Purpose Monitors (SPM). The expected number of exceedances for the year is equal to the observed number of exceedances plus the increment associated with the missing data. The following formula must be used for these computations:

$$e = v + [(v/n)*(N-n)] \quad (1)$$

where

e = the estimated number of exceedances for the year
N = the number of days in the year (365 or 366)
v = the observed number of exceedances
n = the number of valid daily values

* Indicates multiplication.

This expected number of exceedances must be rounded to one decimal place (fractional parts equal to 0.05 should be rounded up).

This same formula is applicable for both 1-hour and 8-hour computations. It basically assumes that the fraction of missing values above the standard level is the same as the fraction of measured values above this level. For 1-hour computations, the observed number of exceedances refers to the hourly standard and the number of valid daily values is the number of valid daily maximum 1-hour averages. For computations for the 8-hour standard, exceedances refer to the 8-hour standard and the number of valid daily values is the number of valid daily maximum 8-hour averages.

(42 U.S.C. 7409)

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40 CFR Part 50

[FRL 1567-4 Docket Number OAQPS 79-7]

Proposed Revision of The National Ambient Air Quality Standard for Carbon Monoxide; Public Meeting Schedule

AGENCY: Environmental Protection Agency.

ACTION: To announce public meetings.

SUMMARY: In this issue of the **Federal Register** EPA has proposed revisions to the National Ambient Air Quality Standard for Carbon Monoxide. This notice is to announce public hearings to be held in Washington, D.C., and Denver, Colorado, to receive public comments on the proposed revisions to the standard. All hearings begin at 9:00 a.m.

DATES: Public comments on the proposed revisions must be received by November 10, 1980.

Hearings will be held on October 2, 1980 in Washington, D.C., and on October 10, 1980 in Denver, Colorado.

ADDRESSES: Public comments must be submitted (duplicate copies are preferred) to: Central Docket Section (A-130), Environmental Protection Agency, Attn: Docket No. OAQPS 79-7, 401 M Street, SW, Washington, D.C. 20460.

The hearings will be held in the following locations: Washington Hearing: Environmental Protection Agency, 401 M Street, SW., Room 3906 and 3908, Washington, D.C. Denver Hearing: U.S. Post Office Auditorium, 1823 Stout Street, (Denver, Colorado). For further details contact Mr. Charles

Stevens, telephone (303) 837–5927 (FTS 327–5927).

Individuals making prepared statements at the hearing are encouraged to send written copies of their statements seven days prior to the day of the hearing to the address given below.

FOR FURTHER INFORMATION CONTACT:

Mr. Michael Jones, Strategies and Air Standards Division, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, N.C. 27711, Telephone (919) 541–5231 (FTS 629–5231).

Dated: July 16, 1980.

David G. Hawkins,

Assistant Administrator for Air, Noise, and Radiation.

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